Case Report Rapport de cas

Long-term outcomes following plate stabilization to address spontaneous luxation of the long digital extensor tendon of origin in 2 dogs

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Abstract — Two dogs with spontaneous luxation of the long digital extensor tendon of origin were managed by performing a sulcoplasty and applying a plate bridging the extensor sulcus. Lameness resolved and neither dog had recurrence of lameness 59 and 15 months following surgery.

Résumé — Résultats à long terme après la stabilisation par plaque pour régler la luxation spontanée de l'extenseur antérieur des phalanges d'origine chez 2 chiens. Deux chiens souffrant d'une luxation spontanée du tendon du muscle long extenseur des doigts ont été gérés en réalisant une sulcoplastie et en appliquant une plaque reliant le sulcus de l'extenseur. La boiterie s'est résorbée et ni l'un ni l'autre des chiens n'a eu de récurrence de boiterie après 59 et 15 mois après la chirurgie.

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The long digital extensor (LDE) muscle has its origin in the extensor fossa on the lateral condyle of the femur (1–7). The tendon of origin, which is encapsulated in a synovial sheath, courses distally, traversing the stifle and extending into the extensor sulcus located on the craniolateral aspect of the proximal tibia (2–6). The tendon is maintained in the sulcus by a thin, retinacular-like restraining band of fibrous tissue (3,4,8). An osseous protuberance, referred to as the tubercle of Gerdy in humans (9), forms the caudolateral margin of the extensor sulcus and functions to maintain the position of the tendon in the sulcus (6,9). The spindle-shaped muscular portion of the LDE is positioned between the cranial tibial muscle and the fibularis longus muscle on the craniolateral aspect of the crus (5). The muscle traverses the length of the tibia with the distal tendon dividing into 4 branches near the tarsus and inserting on the third phalanx of digits II through V (1,3,5,7). The LDE muscle functions primarily to flex the tarsocrural joint and to extend the digits (1,3–6).

Spontaneous luxation of the LDE tendon of origin is an uncommon cause of pelvic limb lameness in dogs (2,3,6,10). Although spontaneous luxation of the tendon of origin of the LDE and treatment options are described in numerous textbooks (2,6,7,10–13), there is limited specific case information regarding the clinical abnormalities, surgical treatment intricacies, and clinical outcomes of dogs affected with this condition (1,3,8). The objective of this report is to describe the presenting clinical abnormalities and surgical management utilizing plate stabilization to maintain reduction in 2 dogs with pelvic limb lameness ascribed to spontaneous luxation of the LDE tendon of origin. Direct long-term functional outcome was obtained in 1 dog and indirect long-term outcome was obtained for the second dog.

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Case description

Two dogs were referred for evaluation of left pelvic limb lameness. Dog #1 was a 6-year-old, 14.2 kg, castrated male Pembroke Welsh Corgi. Dog #2 was a 3-year-old, 12.7 kg, spayed female Shetland sheepdog. There was no known history of trauma or an inciting incident in either dog. The duration of lameness was 1 mo for Dog #1 and 3 mo for Dog #2. Dog #1's lameness improved with exercise restriction and administration of carprofen (Rimadyl; Zoetis, Parsippany, New Jersey, USA), 1.8 mg/kg body weight (BW) q12h, but did not resolve completely. Dog #2 had not received treatment before referral.

On presentation, Dog #1 had a moderate persistent weight-bearing lameness at a walk and a trot. Dog #2 was not lame while walking, but shifted more weight to the right pelvic limb when standing. Both dogs intermittently maintained the affected limb off the ground in flexion while trotting. Dog #2 had mild palpable atrophy of the quadriceps muscles and caudal thigh musculature. Intermittent luxation of the LDE tendon of origin caudolateral to the tubercle of Gerdy was elicited on palpation in both dogs: obvious visual and palpable cranial and caudal movement of the tendon was detected during flexion and extension of both dogs' left stifle.

Both dogs were sedated and bilateral stifle radiographs were obtained. Mild effusion and mild osteoarthritis were noted in

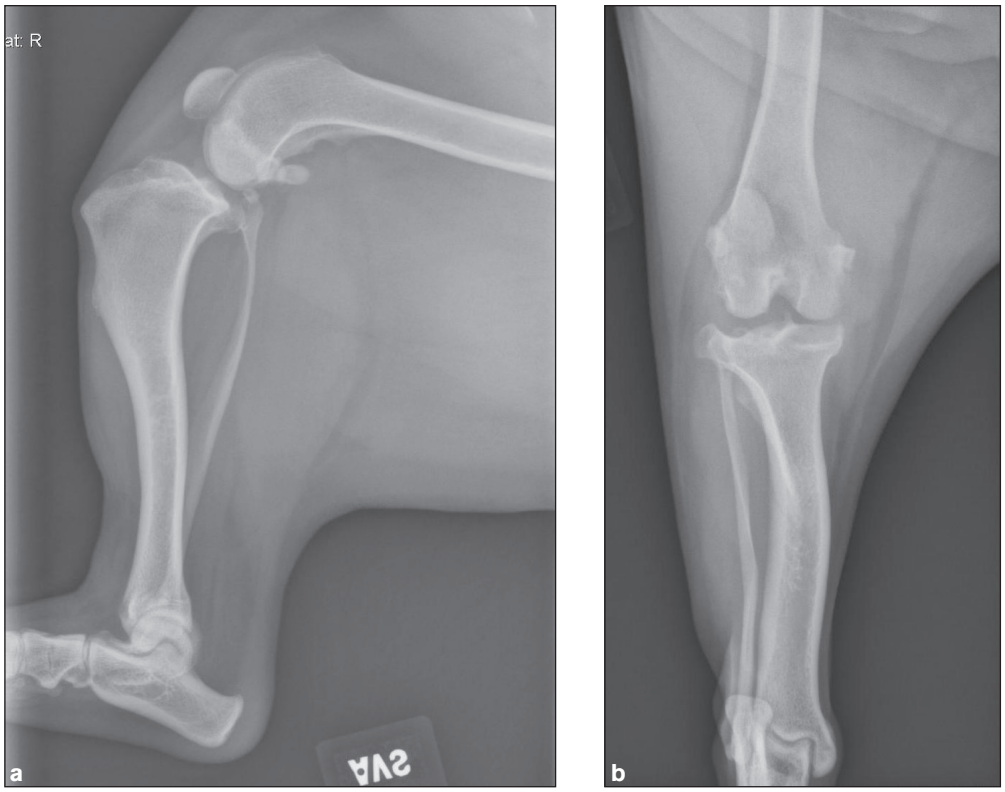

Figure 1. Lateromedial (a) and craniocaudal (b) radiographs of Dog #1 showing the affected stifle during the dog's initial evaluation for lameness. There is mild effusion and early osteoarthritis of the left stifle.

the left stifle of Dog #1 (Figure 1). The right stifle was normal. Dog #2 had moderate effusion and mild osteoarthrosis of the left stifle. The right stifle had mild degenerative changes, but there was no effusion. Pelvic radiographs were unremarkable in both dogs. Neither dog had appreciable medial buttress, patellar luxation, cranial tibial thrust, or cranial drawer when palpated under sedation.

Both dogs were anesthetized and a standard left lateral parapatellar arthrotomy was performed (14). The cranial and caudal cruciate ligaments as well as the cranial poles of the menisci were normal. Mild osteophyte formation was present along the abaxial surfaces of the femoral trochlear ridges and at the origin of the LDE tendon in both dogs. The LDE tendon of origin was edematous and inflamed with neovascularization. The LDE tendon of origin luxated caudal to the extensor sulcus when the stifle flexed in both dogs (Figure 2). The tendon returned to the sulcus when both dogs' stifles were extended, but the tendon did not seat normally within the sulcus due to fibrous proliferation within the extensor sulcus. Fibrous, proliferative synovial tissue enveloped the LDE tendon of origin in Dog #2 and the band of fibrous tissue which normally restraints the tendon in the sulcus could not be identified in either dog.

The proliferative fibrous tissue surrounding and lining the extensor sulcus was removed with rongeurs and a groove sulcoplasty was carried out using a 4-mm bone rasp. The sulcoplasty allowed for normal seating of the tendon below the prominence to the tubercle of Gerdy. Following sulcoplasty, stifle flexion and extension no longer resulted in luxation of the tendon. With the LDE tendon of origin repositioned in the sulcus, a 3-hole, 2.0-mm dynamic compression plate was contoured and applied, bridging the proximal extent of the extensor sulcus. The plate was secured to the proximal tibia using one 2.0-mm screw placed cranial and one 2.0-mm screw placed caudal to the sulcus (Figure 3). The joint capsule and fascia were closed in a simple continuous pattern using 2-0 PDS (Ethicon, Sommerville, New Jersey, USA). Subcutaneous tissues were opposed using 3-0 Monocryl (Ethicon). Skin was closed using 4-0 Monocryl (Ethicon) in an intradermal pattern. Stifle radiographs confirmed proper implant positioning (Figure 4). Dog #2 was administered an injection of cefovecin (Covenia; Zoetis), 8 mg/kg BW, SC after surgery.

Both dogs were discharged the day following surgery and the owners were instructed to administer carprofen (Rimadyl; Zoetis,), 2.2 mg/kg BW, q12h for 7 d, and tramadol (Amneal Pharmaceuticals, Hauppauge, New York, USA), 4 mg/kg BW, every 8 to12 h as needed for pain. Dog #1 was also prescribed cefpodoxime proxetil (Simplicef; Zoetis), 10.6 mg/kg BW, q24h for 8 d. Discharge instructions included exercise restriction, which consisted of confining the dog to a crate or small room when unattended and short walks outside on a leash mainly for urination and defecation.

The incisions had healed without complications when both dogs were re-evaluated 2 wk after surgery. Each dog was using the limb consistently at a walk and trot, with only a mild persistent weight-bearing left pelvic limb lameness. The stifles were comfortable on palpation and the LDE tendon of origin did

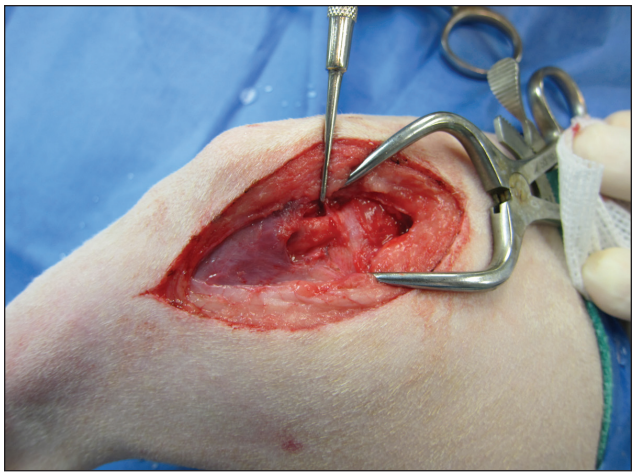

Figure 2. Intra-operative photograph of Dog #1's left stifle. The ball end probe is pointing to the inflamed, caudally luxated LDE tendon of origin.

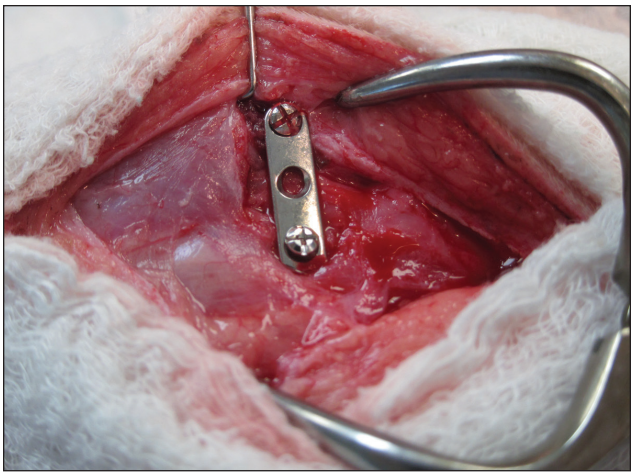

Figure 3. Intra-operative photograph of Dog #1's left stifle following plate contouring and placement. The LDE tendon of origin is reduced within the sulcus extensorius.

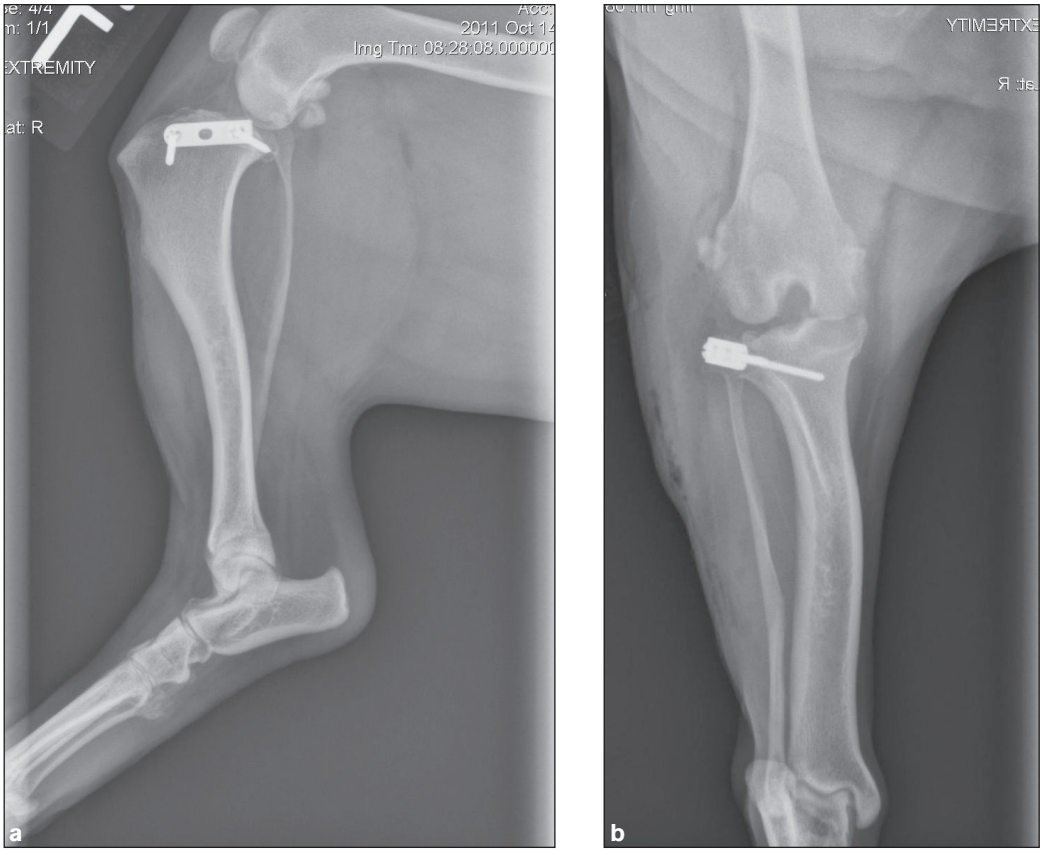

Figure 4. Lateromedial (a) and craniocaudal (b) initial post-operative radiographs of Dog #1. The radiographs revealed appropriate implant positioning.

not luxate as the joint was moved through a range of motion. Neither dog exhibited lameness when examined at 4 and 8 wk after surgery. The LDE tendon of origin did not luxate as either dog's left stifle was flexed and extended. Radiographs of the left stifle showed that the position of the implant was unchanged in each dog. No complications were noted in association with the surgery. Improvement of the previously noted effusion with static degenerative changes was noted on the follow-up radiographs. A gradual return to normal activity was recommended after the 8-wk post-operative evaluation. Dog #1 was also evaluated 12 wk after surgery. No lameness was noted and palpation of the left stifle was unremarkable.

Dog #1 was re-examined 5 y following surgery to obtain long-term results (15). The owner reported a normal level of activity without lameness. During the orthopedic examination, lameness was not observed at a walk or a trot. Pain was not elicited on hyperextension of either stifle. Neither stifle had palpable effusion, medial buttress, or crepitus. Luxation of

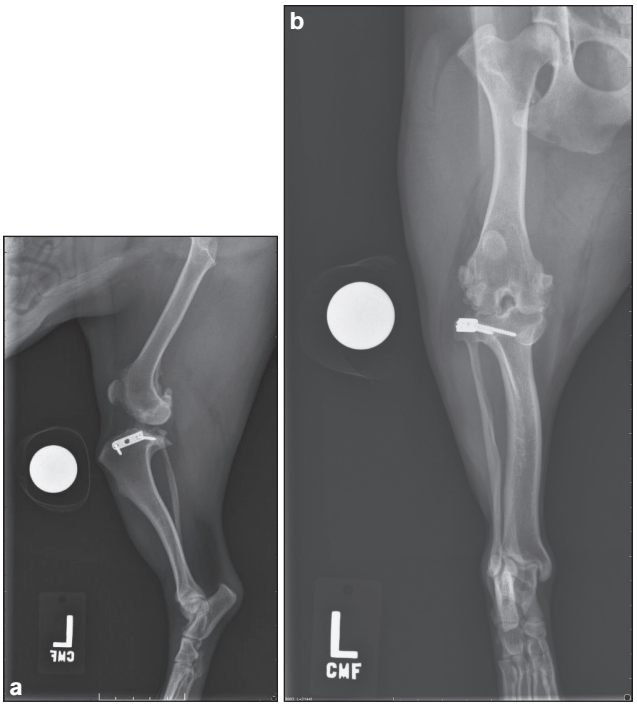

Figure 5. Lateromedial (a) and craniocaudal (b) long-term follow-up radiographs of Dog #1 obtained 5 y after surgery. Radiographs revealed mild effusion with moderate to severe osteoarthrosis associated with the femoral trochlear ridge, medial and lateral femoral epicondyles, fabellae as well as medial and lateral tibial condyles. There was remodeling of the tibial plateau and intercondylar eminences. The implants were unchanged in position compared with previous radiographs.

the LDE tendon of origin could not be elicited as the left stifle was moved through a range of motion. The dog maintained a normal, square posture at a sit. The dog was walked over a force platform (Model# OR6-6-1000; Advanced Mechanical Technology, Newton, Massachusetts, USA) and there was mild asymmetry between the pelvic limbs (Table 1). The dog was sedated and radiographs of both stifles were obtained. The left stifle had mild effusion with moderate osteoarthrosis, principally affecting the lateral compartment of the joint (Figure 5). Mild coxofemoral osteoarthosis was noted bilaterally. While the dog was sedated, pelvic limb circumference (Gulick tape measure; manufacturer unknown, Vietnam) and joint flexion and extension angles (Goniometer; IMEX Veterinary, Longview, Texas, USA) were measured (Table 1). Palpable bilateral coxofemoral laxity was detected, but neither patella could be luxated. Cranial tibial thrust and cranial drawer could not be elicited on examination under sedation. Direct long-term evaluation was not available for Dog #2; however, the owner was contacted 15 mo after surgery and reported that the dog had not had any recurrence of lameness and was currently being used to herd sheep.

Discussion

Spontaneous LDE tendon luxation has only been previously reported in 2 dogs, 1 of which was affected bilaterally (1,3). A third dog, reported by Bennett and Campbell (8), had a mal-union femoral fracture, which resulted in femoral varus and coxofemoral joint incongruency: these conformational abnormalities or the inciting trauma presumably contributed to this dog developing luxation of the LDE tendon of origin. Long digital extensor tendon luxation has also been reported as a consequence of tibial plateau leveling osteotomy (4) and tibial tuberosity transposition (16). The condition has also purportedly been associated with patellar luxation (2). Etiology for spontaneous luxation of the LDE tendon of origin is unknown (1,3), and no underlying cause has been proposed for this condition (2,6). At the time of diagnosis, the dogs herein were of similar age to the dogs in previous reports (1,3). There are not enough reported cases to determine if there is an age, gender, occupation, or breed predilection for spontaneous LDE luxation.

The lameness associated with LDE tendon luxation can be variable, ranging from none to a marked lameness with intermittent periods of non-weight-bearing lameness (1–3,6,10–12). In the current report, Dog #1 had a persistent weight-bearing lameness at a walk, whereas Dog #2 was not lame while walking, but shifted more weight to the contralateral pelvic limb when standing. Both dogs intermittently held the affected limb up in flexion when trotting.

Consistent with prior descriptions (1–4,6–8,12), a popping sensation was detected in both dogs as the tendon luxated and reduced during stifle flexion and extension. The popping can be audible and visually recognized in shorthaired dogs or dogs in which the hair has been clipped over the stifle (1,3,8). The popping sensation can be confused with a meniscal click or patellar luxation (2,17). Neither of the dogs reported here had a patella luxation or instability associated with cranial cruciate ligament insufficiency on palpation. Both dogs had radiographic evidence of stifle effusion and degenerative changes which could be consistent with cranial cruciate ligament disease. The cruciate ligaments and the visible portions of the menisci were found to be normal at surgery. Lameness in both dogs resolved after the LDE luxation was addressed and neither dog developed lameness, thrust, or drawer following surgery. Similar radiographic effusion and degenerative changes have been reported and were ascribed to inflammation produced by the intermittent luxation of the LDE tendon of origin (3).

At surgery, the band of tissue that normally restrains the tendon within the sulcus was unidentifiable in both dogs. Displacement of any tendon requires disruption of the tendon's restraining connective tissues (4,7,8). Neither of these cases had a history of trauma. Of the 3 reported cases (1,3,8), only 1 dog had a known history of prior trauma (8). We suspect that traumatic disruption of the restraining band was the underlying cause of luxation in our cases, as the restraining fibrous band was not identifiable at surgery.

Pathology of the extensor sulcus was not described in previous reports of dogs affected with LDE tendon luxation (1,3,8), but a sulculoplasty was performed in these dogs to remove proliferative fibrous tissue and address remodeling which limited effective seating of the tendon within the sulcus. A shallow sulcus can be a consequence of osseous proliferation (4) and a bone rasp was used to increase the depth of the sulcus in our cases. Alternatively, rongeurs or a pneumatic burr could have been used to perform the sulculoplasties (6).

Table 1. Force plate, limb circumference, and goniometry data for Dog #1 obtained 5 y after surgery.

	Force plate data		Limb circumference (mm)		Goniometry (°)					
					Hock		Stifle		Hip	
Limb	PVF 100*N/N	PVI 100*N-s/N	Crus	Thigh	F	E	F	E	F	E
Affected	74.71	6.57	124	248	35	153	54	116	26	130
Contralateral	77.26	6.56	135	258	40	157	45	122	26	126

PVF — peak vertical force; PVI — peak vertical impulse; F — flexion; E — extension.

Previous reports of the surgical stabilization of the LDE tendon describe using a small drill bit or Kirschner wire to create bone tunnels through the cranial and caudal prominences of the extensor sulcus: suture or small gauge wire was secured through the bone tunnels to maintain the tendon in the sulcus (1–4,6–8,10–13). If the protuberances are not large enough to accommodate drilling bone tunnels, sutures can be anchored in periosteum and fascia (2,6). The use of a staple, fashioned from a Kirschner wire, has also been described (4). In our dogs, a contoured, 3-hole 2.0-mm plate was placed over the sulcus and secured with 2 screws to provide a permanent restraint to maintain reduction of the tendon. The use of a plate and screws reduces the risk of recurrence secondary to suture breakage or pull through or failure of a small diameter staple and does not require suitable anchorage points adjacent to the sulcus to accommodate bone tunnels. This technique was simple and effective as documented at the time of long-term evaluation in Dog #1.

Reduction of the LDE tendon luxation was maintained following surgery in both dogs reported here as well as in previously reported cases (1,3,8). Our favorable perioperative outcomes (15) were similar to previous reports (1,3); however, limited details and follow-up evaluations were available in previous reports (1,3). Both of the dogs reported here had mild weight-bearing lameness 2 wk following surgery. Luxation of the LDE could not be elicited on palpation. Neither dog was lame at subsequent examination. Radiographs obtained at 1 and 3 mo following surgery revealed no complications associated with the implants, decreased stifle effusion, and static degenerative changes.

Direct long-term outcome (> 12 mo) assessment (15) was obtained for Dog #1: the dog was sound on the affected limb when both walking and trotting and the LDE tendon did not luxate during stifle range of motion. On long-term follow-up radiographs for Dog #1, progression of osteoarthritis was more advanced in the left stifle than the right. The degenerative changes were most pronounced on the lateral aspect of the stifle, consistent with the location of the origin and the tract of the LDE tendon within the extensor sulcus. There was also remodeling of the intermediate ridge of the tibia and proliferative osteophytes on the lateral femoral condyle. Objective measures of limb function revealed nominal differences between pelvic limbs, which may be attributable to the degenerative changes noted on radiographs. Despite these degenerative changes, Dog #1 continued to have normal use of the affected limb. Although we were not able to conduct a direct long-term outcome assessment for Dog #2, the owners did not perceive that their dog had any lameness and they reported that the dog was actively working on their farm. These positive long-term results validate the efficacy of our surgical technique for this uncommon cause of pelvic limb lameness in dogs.

Acknowledgment

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